Section of Pædiatrics

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[*April* 27, 1951]

DISCUSSION ON THE TREATMENT OF CONGENITAL HYPERTROPHIC PYLORIC STENOSIS

The President: Infantile hypertrophic pyloric stenosis is a common disease—occurring once in approximately 250 live births, and as its treatment is still controversial it is surely a matter well worth further discussion.

Until the close of 1944 I, myself, had always regarded the treatment of infantile hypertrophic pyloric stenosis as essentially surgical, and just before the last war I published a report of a series of 102 cases, 98 of which had been submitted to operation (Tallerman, 1938). With the exception of three or four, all of these 98 cases had been operated on by one or the other of my colleagues, Sir James Walton or Mr. David Levi; the mortality in this series was 14%. This mortality rate was about the same as that of other large series of cases reported at that time. If one adds to my series of 98 (1938), the 209 cases reported by Thompson and Gaisford (1935), and the 303 recorded by Jewesbury and Page (1937), a total of 610 cases treated surgically is obtained, of which the mortality was 13·4%. Certainly since then there have appeared reports in which the mortality of cases operated on is far lower. In a recent paper, Grimes, Bell, and Olney (1950) of California state that between 1930 and 1950, 113 patients suffering from this condition were operated upon without a fatality; in 81% a tumour had been felt. In this country Levi (1941) has also published a series of 100 consecutive cases occurring in breast-fed infants who were operated on without a single death. On the other hand, in the same paper he records a mortality of approximately 11% of 46 artificially fed infants submitted to operation on account of this disease, and he quite rightly stresses the important influence of breast feeding on the prognosis.

We have, however, always to reckon with infants suffering from pyloric stenosis who happen to be artificially fed, and as this disorder is so common it seems to me to be a mistake to fix attention too closely on the results obtained by any particular surgeon in any particular centre, where special skill, experience and facilities all combine to produce particularly good results. I believe that a general survey of the surgical treatment of pyloric stenosis in this country would reveal the fact that a mortality rate of about 15% at various centres was not uncommonly high. What seems to me to be striking is the lower mortality rates reported even many years ago in a large series of cases treated by medical means alone. For example, Faxén (1933), without mentioning diagnostic criteria, reported 126 cases with a mortality rate of 5·5%. Exactly the same mortality occurred among 179 cases recorded by Svensgaard in 1935 (a pyloric tumour being felt in 87%), and it was the publication in this country of her paper that gave an impetus to the use of Eumydrin (a proprietary preparation of atropine methylnitrate), and the medical treatment over here of pyloric stenosis in infancy. Further reports on the treatment of this disorder by medical means alone have since been published, and the literature on this whole subject is very considerable.

Since the autumn of 1945, I have deliberately set out to treat every case of pyloric stenosis by medical means. If the patient has failed to respond satisfactorily and symptoms have persisted I have then resorted to surgical treatment. In order to ensure that an infant is not in poor condition and therefore a bad operative risk I am, generally speaking, in favour of seeking surgical help if there is still vomiting and failure to gain weight after about seventy-two hours from the beginning of treatment.

Atropine methylnitrate is given either as lamellæ, in aqueous solution, or in a 0·6% alcoholic solution; in addition stomach washing with saline is carried out, twice daily as a rule for the first three or four days, and saline is given subcutaneously if there is any evidence of dehydration. The feeding régime is also suitably adjusted as may be necessary.

The cases on which I now wish to comment comprise a series of 67 treated in this fashion; they all occurred between November 1, 1945, and October 31, 1950, and 6 of them died. In only one of these cases was a pyloric mass not felt on some occasion while the child was under treatment, and in this patient, who actually came eventually to operation, the diagnosis was confirmed at that time. In all other patients operated upon, the diagnosis was also then confirmed in every case. In 5 cases, although a pyloric mass was felt, visible peristalsis was not observed. Now of these 67 patients, 41 (61%) were treated medically throughout with a satisfactory result in 40 cases. Of these, 4 were never admitted to hospital. 26 patients (39%) were submitted to operation owing to their failure to respond satisfactorily to medical treatment alone. This may have been because the dose of atropine methylnitrate was not pushed sufficiently. Looking back on this series I regard 5 of the 26 cases who were

operated on as having received too small a dose of atropine methylnitrate during their period of medical treatment. I now use a 0·6% alcoholic solution of this drug, and consider that nothing less than 15 drops daily, given in divided doses before each feed, is adequate, and that if a satisfactory response is not obtained this should be rapidly increased to 25 drops. That is to say, 1/50 of a grain of the drug daily is the minimum dose which should be prescribed. I suspect that disappointing results of this treatment have sometimes been due to a failure to push the drug sufficiently.

What seems to be impressive about this series of cases is the fact that of the total number nearly two-thirds responded satisfactorily without being submitted to operation. Still more important is the fact that, while of the 41 treated medically only 1 died, of the 26 operated on no less than 5 died; a mortality rate of approximately 2½% against 19%. Now it may be argued that those who required operation were more seriously ill and in worse condition than those who were not operated upon. I am not sure of this, though I admit that of the 26 cases operated on 11 were recorded on admission as being in poor general condition, while of the 41 cases treated by medical measures throughout, the condition of 6 was recorded as being poor. Judged on a weight basis, the infants operated on did not seem to have deteriorated between the time of admission and the day of resorting to operation, as their average loss in weight was little more than half an ounce during this period of treatment. In regard to the infant treated medically who died, he was in hospital for two weeks, but after discharge unfortunately failed to receive atropine methylnitrate at home. He was subsequently readmitted to hospital in poor condition and died of gastro-enteritis.

No child over 2 months of age at the time of admission required operation, and, with one exception, all those operated on had presented symptoms for a period of no longer than a month, the average (excluding this one exception) being thirteen days. This may, perhaps, be thought to indicate that patients in whom symptoms have persisted for some time and who are first seen at 5 or 6 weeks of age are those in whom the condition is more amenable to pure medical treatment; this, however, is rather speculative, for in those of this series who required medical treatment alone the average duration of symptoms was nineteen days, which was only six days longer than the average of those who came to operation.

One thing is quite clear, and that is that approximately two-thirds of all cases responded satisfactorily to medical treatment alone, and that the difference in mortality between those so treated and those surgically treated is most striking.

In these circumstances surely it is wise to give an infant the benefit of medical treatment in the first instance provided surgical aid is sought if he fails to respond readily to medical treatment alone. It has been argued that medical treatment entails a long period as an in-patient, with the consequent risk of cross-infection. This is not so, and some infants can be treated as out-patients without being admitted to hospital at all; others, after a short period of in-patient treatment, can be discharged from hospital and attend as out-patients. In this series the average duration of stay in hospital was thirteen days for those who were operated on and twenty days for those treated exclusively by medical means, a difference of only seven days. In assessing these figures the cases treated entirely as out-patients have, of course, been excluded (*see* Table I). It should be possible to reduce still further the

TABLE I

HYPERTROPHIC PYLORIC STENOSIS OF INFANTS

67 cases (during the five-year period 1.11.45 to 31.10.50)
6 deaths (9% mortality)

Of these 67 cases:
- 41 treated by medical means alone (61%)
  - 1 death (2·5% mortality)
- 26 treated surgically (39%)
  - 5 deaths (19% mortality)

AVERAGE LENGTH OF STAY IN HOSPITAL

37 cases treated medically as in-patients	20 days
26 cases treated surgically as in-patients	13 days

4 cases were treated medically without ever being admitted to hospital.

(It is realised that percentages may be misleading when so few cases are involved, but they are given simply as a convenient basis of comparison with other results.)

period of hospital treatment, and it is our aim to do so. Of the infants in this series treated medically nearly two-thirds were artificially fed; of those treated surgically, the numbers breast-fed and artificially fed were about equal, but all those who died after operation had been fed artificially.

Apart altogether from the published evidence of the efficiency of medical treatment, support for which is also to be found in this present series, there seems to be another way of viewing the matter. No one, I hope, regards congenital pyloric stenosis as a true surgical emergency, and all would surely agree as to the importance of preoperative preparation of the patient destined for surgical treatment: the use of gastric lavage, administration of subcutaneous saline when necessary, and careful feeding.

Why not then during this preoperative period of two to three days also prescribe atropine methylnitrate in adequate doses before each feed? If symptoms cease and the infant gains weight he can usually be discharged in a short time from hospital, and continue treatment as an out-patient. If he fails to respond he can and should then be operated on without delay. If this line is adopted, roughly two-thirds will escape what is a major abdominal operation, associated with a far from negligible mortality.

It is quite erroneous to state that the treatment of congenital pyloric stenosis is necessarily either purely medical or purely surgical. Some medical treatment is always necessary, and surgical intervention may subsequently be required.

I should like to thank those of my colleagues who have allowed me to include in this series patients that were under their care, and particularly Dr. Nauth-Misir for his assistance. I am also indebted to Dr. H. G. Dunn for helping to assemble the data for this paper.

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Mr. Denis Browne: To reduce the mortality of pyloric stenosis to the minimum the important cases to consider are the very ill babies. These need a full feed with the least possible delay, which can only be assured by operation. Like all starved patients they take general anæsthetics badly, and so benefit by the use of local anæsthesia instead. It appears to me reasonable to attain proficiency in the far from easy technique most suitable to the weakest by applying it to all cases, including the strongest. I think the advantages of local anæsthesia are obscured in statistics by its reservation for the weakest babies, and its use on them by surgeons who are not familiar with it.

TECHNIQUE OF RAMSTEDT'S OPERATION UNDER LOCAL ANÆSTHESIA

(1) *Preoperative treatment.*—A valuable temporary improvement can be gained by giving food and fluid intravenously, washing the debris of previous feeds out of the stomach, and getting the blood chlorides as near normal as possible.

(2) *Premedication.*—Two grains of chloral two hours before operation.

(3) *Fixation.*—A padded wooden frame or "crucifix" keeps the baby still and warm.

(4) *The anæsthetic.*—0·5% novocain with adrenaline, from a freshly opened bottle made up by one of the great wholesale firms. It is safe to use up to 15 c.c.

(5) *The area injected* should be that shown in Fig. 1. Skin and muscles should be injected separately. Five minutes is allowed for the solution to act before making the incision.

(6) *The incision.*—A vertical split of the right rectus, exposing the liver only.

(7) *The "bootlace stitch".*—The reason why surgeons give up operations under local anæsthetic is usually one: the difficulty of closing the peritoneum. It is easiest to put in a stitch for this before the child has been annoyed by the dragging out of its stomach. Fig. 2 shows how this can be done as soon as the peritoneum is opened. This stitch must be tightened at the end of the operation loop by loop, picking them up in succession by forceps.

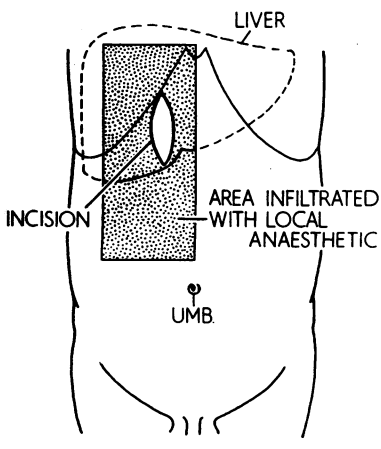

FIG. 1.

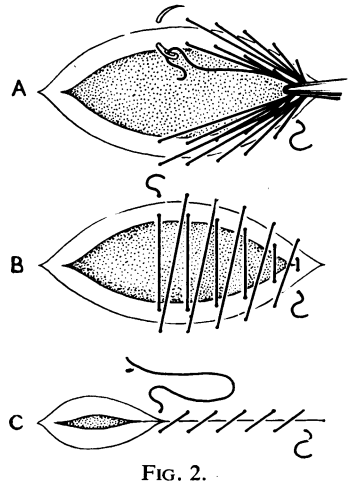

FIG. 2.

FIG. 1. — Diagram of infiltration and incision.

FIG. 2. — Insertion of "boot-lace stitch". A, The stitches being inserted, each one being looped round an artery forceps gripping the lower angle of the wound. B, Appearance when the artery forceps has been removed after the replacing of the stomach in the abdomen. C, Appearance after the stitches have been tightened up, each one separately. The first stitch should have been tied first.

(8) *Deflating the stomach.*—The occasional baby who struggles when the anæsthetic is properly given does so, not because he is feeling pain, but because his stomach is uncomfortably distended with air. Many of these babies have the trick of swallowing air, and some of them in this way get up an almost incredible pressure; I have seen the stomach fly out of the incision like an inner tube out of a burst motor tyre. In consequence, there should always be a stomach tube ready, with someone capable of passing it; and it should be used without delay if the stomach is felt to be unduly full. Inflation beyond a certain degree also makes it very hard to bring the stomach out through a small incision.

(9) *Finding the pylorus.*—The pyloric tumour cannot be picked up directly through the incision recommended. The liver should be pulled upward with the left forefinger, and the stomach will often then appear under its edge, when it can be picked up by that instrument with a blunt overlapping grip known as my tonsil-holding forceps. If the stomach does not appear, it is easy to find the omentum, if necessary by pushing into the abdomen a piece of gauze, to which it will invariably adhere. The omentum pulls up the colon, the colon the stomach, and the stomach the pylorus.

(10) *Incision of the pylorus.*—This incision should begin about a ½ cm. short of the junction of the duodenum with the pylorus, and run in a slight curve well up on to the stomach wall. Most illustrations give the idea of too short and straight an incision; and it is important that the muscle towards the point at which perforation is liable to occur, the "zone of adhesion" at the duodenal junction, should be torn apart and not cut. Not everyone has studied the anatomy enough to realize that perforation occurs very superficially, at the end of the fornix round the cervix-like protrusion of the pylorus, and not in the depths of the incision.

(11) *Dividing the muscle.*—This is begun by a semi-sharp dissector, and continued by tearing apart the edges of the wound. My instrument for this has semi-sharp out-turned points that dig into the thick muscular edges.

The sign of complete division is the paradoxical one that the bore of the released mucosa suddenly narrows, thus showing that the "cervix" has been reached. A width of at least half an inch of mucosa must be exposed in the centre of the incision (Figs. 3 and 4).

FIG. 3.—Splitting of the pyloric muscle.

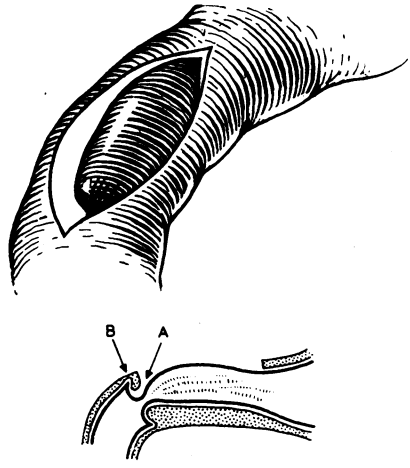

FIG. 4.—Diagram to show A, The "zone of constriction" of the mucosa which is the sign the pylorus is freed, and B, The "zone of adhesion" at which perforation is likely.

(12) *Controlling the escape of small intestine.*—On returning the stomach the omentum should be grasped in artery forceps and thrust downwards into the abdomen. This pulls down the transverse colon and so controls the coils below it.

(13) *Suturing.*—Peritoneum and muscles are sewn up separately by catgut; the skin by interrupted silk sutures. A tight adhesive dressing is put over the wound and two-thirds of the way round the body, being left untouched for eight days. A slight discharge through the adhesive is an indication for leaving it on rather than taking it off.

An objection sometimes made is that operation under local anæsthesia takes too long. On two occasions I have done a list of 4 cases in succession, and in each the 4 were complete in almost exactly two hours. How much less time would have been occupied with a similar list done under general anæsthesia seems to me of very little importance.

RESULTS

In 407 successive unselected cases done by this method during 1943–45 at The Hospital for Sick Children, the mortality remained curiously steady at 2%. All the others left hospital with the

stitches out, gaining weight, and on full feeds. There was no operative mortality, and the ones who died suffered from a variety of handicaps besides the pyloric block. It would have been possible to select a series of 100 cases with 100 recoveries, but that is not the same thing as a 100% recovery rate. Those who have no mortality in this condition must enjoy a curious freedom from cases complicated by such conditions as pneumonia, enteritis, abscesses, or congenital defects.

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Mr. David Levi [*Abstract*]: Success in the surgery of infancy, and especially in pyloric stenosis, depends upon meticulous attention to detail from the moment the baby leaves its mother to the time when it is returned to her.

The safest place for any baby is at home in its own mother's care, provided that the mother is possessed of average intelligence. The most dangerous place for a baby may be with other sick babies in a ward.

These dangers can be minimized by building a hospital or accommodation in a hospital specially for babies, and secondly by training the nursing staff, resident staff and the wardmaids in the care of infants. The problem is to keep the baby free from intercurrent infection and to guard it from ignorant and callous nursing.

Breast-fed babies should be admitted to separate rooms with their mothers. A bottle-fed baby should have a room to itself.

The ward kitchen should be well away from the sluice and bathroom; at opposite ends of the corridor, if there is one. Both ward kitchen and sluice room should be provided with a sterilizer. When the feeding utensils are washed, they should be put straight into a sterilizer and boiled. The same procedure should be applied to the toilet requisites in the sluice room which must contain a sterilizer.

Attention must be paid to the sterilizing of all bedding and blankets, each time the rooms are occupied, and precaution must be taken not to raise dust when the floors are swept. Some form of suction cleaner or oiling the floors answers this purpose. All visitors to the rooms should be banned. Only those who have some duty to perform should be allowed.

Great stress should be laid upon the importance of all staff, wardmaids included, reporting immediately if they feel ill and anyone with a loose bowel action, abdominal pain, a sore throat, a mild cold, a septic finger, should not do duty on the babies' floor. The newcomers, especially, should be requested to report symptoms *before* going on duty.

All entering the babies' abode should be masked to prevent droplet infection. Porters and wardmaids are sometimes overlooked in this connexion. Highly trained skilled nursing is an essential.

An untrained but well-meaning probationer can infect an ill baby in a few moments by doing unthinking things which are done every day with impunity at home to well babies. She may, for instance, change the child, put the teat on its bottle, and feed it without washing her hands.

Changing the baby should be performed by a specially gowned masked nurse. The necessary utensils should be sterilized and laid on a tray. The disposal of the soiled square is important. It should not be washed by the nurses, but placed in a covered receptacle and washed by laundry maids, and sterilized in a special laundry right away from the infants.

The infant should not be fed by the nurse at the time she changes it, but the nurse should leave the room with the soiled tray and change her gown. When the baby is fed, the hands of the person feeding it should be washed immediately beforehand and the sterilized teat should not be touched unless the hands are surgically clean. *Each infant should have its own thermometer kept in its room.*

Breast-fed babies are not nearly so liable to contract gastro-enteritis and it is much safer to admit a breast-fed child and its mother than it is to admit a bottle-fed infant.

If all these facilities are not available there are several compromises which might be considered. The baby may be made the personal responsibility of a sister or nurse who should "special" it.

If the baby has to be admitted to a ward its cot should be placed between that of two older children or between two adults. The children should not be infectious or have running ears. A number of babies should not be admitted to the same ward. Nor should one baby be placed next to another. The nurse who attends to the infants should not wash their soiled napkins.

It is my practice to treat hypertrophic pyloric stenosis as an urgent surgical condition, and to operate upon such children as soon as the condition has been diagnosed. The only pre-operative preparation given is a stomach wash-out. This is to remove decomposing stomach contents and to remove the air.

The operation should be performed using 10 c.c. of 0·5 procaine solution for anæsthesia. This is important in ensuring a speedy post-operative convalescence. *It enables the child to be fed by the mouth three hours after operation and it enables the surgeon to abolish from his mind all consideration of intravenous therapy.*

Standardization can only be achieved by mouth feeding which is the most satisfactory and safest method of restoring fluid protein and electrolyte loss yet known.

A film demonstrating the operative technique was then shown.

There have been no deaths from hypertrophic pyloric stenosis or its complications in a series of 125 consecutive patients. One child admitted with staphylococcal osteomyelitis of the facial bones, also suffering from pyloric stenosis, was operated upon and died fourteen days later. Post-mortem revealed a cerebral abscess, lung abscesses and abscesses in the kidneys. Of the 125 patients, 75 came from the Westminster Children's Hospital from the date of reopening in 1947 to date, and 50 were from the Luton Children's Hospital, treated from 1940–1951.

I should like to make a plea on behalf of those babies who are coming into this world to suffer from pyloric stenosis to ask for early diagnosis and treatment, and also to express a hope that more babies will be breast fed and that their attendants will wash and be clean.

Dr. N. M. Jacoby: The treatment of pyloric stenosis was discussed by this Section in 1941 (*Proc. R. Soc. Med.*, **35**, 49) and opinion then was generally in favour of surgery. Probably much the same holds good to-day. Nobody would deny that cases can be cured medically, but the objection is that there may be failures, and subsequent operation has a higher risk than if it were done earlier. Thus the advocates of surgery advise operation in all cases. This seems to ignore the fact that operation is not entirely devoid of risk, and that many parents do not like operations. Personally I do not advise treating all cases medically, because as a universal treatment I think it increases the risk, and nowadays mortality rate should be very low; but it is possible to recognize clinically cases where medical treatment will almost certainly succeed, and even if it should fail, operation will not carry an increased risk. I hope to show that, in this type of medical treatment, cure is just as rapid as surgery and that hospital accommodation is needed for an even shorter period (Fig. 1). The cases not selected for medical treatment are operated on immediately, though I know that some could be cured medically. I am not prepared to run the risk of finding out which they are.

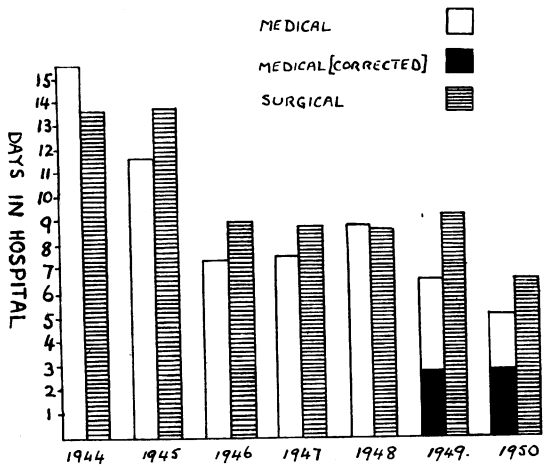

FIG. 1.—Showing the average time spent in hospital by medical and surgical cases from 1944 to 1950. "Corrected Medical" is the average time spent by medical cases, taking into account the extra days where medical treatment failed, and the days saved when the infant was treated at home. (This procedure only started in 1949.)

Selection of cases for treatment is according to the following criteria:—

SURGICAL TREATMENT

(1) Vomiting beginning in the second week or earlier.
(2) Severe dehydration.
(3) Hæmatemesis.
(4) Infant below its birth weight.

MEDICAL TREATMENT

(1) Vomiting beginning in the fourth week or later.
(2) Vomiting continuous for three weeks or more before the infant is first seen, provided it is not severely dehydrated.

Usually the line of treatment is clear, but some cases fall partly into both groups. Here clinical judgment must decide, and if in doubt it is probably wisest to operate.

TREATMENT OF MEDICAL CASES

Feed.—Breast milk or artificial feed 20 calories per oz.
Amount.—Under 6½ lb. 1 oz. four-hourly. Over 6½ lb. 1½ oz. Increase feed by ½ oz. every twenty-four hours.
Method.—Feed in cot for first five days.
Atropine methylnitrate 1/100 grain before alternate feeds. (Lamellæ (Savory & Moore) or 0·6% alcoholic solution.)
No parenteral fluids and no stomach washouts.

RESULTS

Total cases	120 (102 males, 18 females)	
Medical treatment	61 cases	51%
Surgical treatment	59 „	49%
Failed medical	4 „	6·5%
Surgical + Failed medical	63 „	52·5%
Medical cases treated at home	15	
Medical cases failed at home, responded in hospital	2	
Medical cases failed at home. Operation	1	
Deaths	None	

With this treatment vomiting usually stops immediately, and in successful cases never returns. Failure is recognized by a return of serious vomiting on the third to fifth day, whereupon operation should be undertaken immediately. It should be noted that as no stomach washouts or parenteral hydration is used, there is no need for skilled nursing or hospitalization. Provided the mother is reasonably capable the infant can be treated at home.

Treatment of Surgical Cases

In this series almost all cases were treated by the Levi method, with immediate operation under local anæsthesia and without pre-operative hydration except in the severest cases. In the post-operative treatment I have now abandoned all charts and routines. Each case is judged on its merits. In general, feeding starts four hours after operation, and full fluid intake is reached eight hours later. Full caloric intake is reached in from one to seven days according to estimated loss of tolerance for food. If the infant is artificially fed I use half-cream dried milk.

Dr. George Davison quoted as his authority a consecutive series of 1,100 cases of pyloric stenosis treated in Newcastle upon Tyne between September 1925 and April 1951. Of the first 200, treated between September 1925 and January 1936, 55 died (27·5%); out of the last 900 there were 42 deaths (4·7%). More than half of the cases had been supervised personally. In a disease in which the mortality is low great care must be taken in interpreting statistics, and conclusions should not be drawn from small series, nor from numbers of consecutive cases without deaths. Thus, in the Newcastle series, deaths in the last five consecutive hundreds were 1, 6, 2, 3 and 3. It would be true, but misleading, to state that between April 1948 and September 1949, 132 consecutive cases were treated without a death, while 226 consecutive Ramstedt operations were performed without a death between December 1947 and January 1950. The true state of affairs is represented by the average mortality of 3·0% (± 1·5%) over the last 500 cases.

While the mortality in other large pædiatric centres is probably similar to that in Newcastle, the same cannot be said for the country as a whole. Assuming an incidence of 3 to 4 per 1,000 live births it can be calculated that, as there were 679,937 live births in England and Wales in 1945, there must have been 2,000 to 2,700 cases of pyloric stenosis. Of these 286 died, giving a mortality of 10·6% to 14·3%. The mortality outside the larger centres may well be 15% to 20%. Our first step to lower the mortality of pyloric stenosis should therefore be to ensure that all cases come into the hands of those expert in their treatment, whether this skill lies in medical or surgical treatment, or in a combination of both.

Out of the last 15 deaths in the Newcastle series 2 were considered inevitable in view of other deformities, 1 was not understood, 1 was a direct result of operation, 1 was from ward infection; the remaining 10 died because diagnosis had been delayed too long, 4 dying within a few hours of admission. The most important step which will lead to lowering the mortality of pyloric stenosis is therefore education of the general practitioner to ensure that diagnosis will not be delayed. It must be remembered that the general practitioner, seeing on an average only one case every five or six years, cannot be expected to be an expert in the diagnosis of pyloric stenosis, and that, therefore, the feeling of a pyloric tumour, however important it may be in hospital medicine, plays little or no part in diagnosis in general practice. The student and practitioner should be taught to recognize vomiting due to underfeeding and mismanagement of breast or bottle technique; when vomiting occurs in the absence of mismanagement or despite its correction the general practitioner should be encouraged to seek pædiatric help.

Dr. Richard H. Dobbs: Of greater moment than the results of surgical versus medical treatment of pyloric stenosis is the success with which any treatment is carried out in the country as a whole, particularly in centres away from the limelight of published work with long series of successfully treated cases.

The only report that I know of in which this was attempted, and which had this discussion as its objective, was, surprisingly enough, that by Ramstedt himself (1934). Over a long period of time and from a great number of hospitals he collected cases which had been treated on the one hand surgically by the operation that bears his name, and on the other by a considerable variety of medical methods, in which atropine, though not Eumydrin, was often used.

The following table is taken from his report:—

Years	Cases		Mortality%
1919–28	Surgical	497	22·5
	Medical	1,345	16
	Total	1,842	17·9
1929–33	Surgical	757	14·4
	Medical	1,675	9
	Total	2,432	10·7
	Total Surgical	1,254	17·7
	Total Medical	3,020	12·2

Ramstedt's figures suggest not only that even twenty years after his technique was the only one employed throughout the world, medical treatment was still commonly practised; but also that its results were on the whole superior to those from surgery.

The incidence of pyloric stenosis is known, and it is possible, as was suggested by Davison (1946), to get an accurate idea of the mortality from pyloric stenosis in the country as a whole by reference to the Annual Reports of the Registrar-General. Wallgren (1941) assessed the frequency in Gothenburg to be about 4 per 1,000 live births, and, by similar methods, Davison obtained a frequency of almost 3 per 1,000 live births in Newcastle. By multiplying the frequency by the number of live births the total number of cases of pyloric stenosis occurring in the country each year can be quite accurately assessed. The broken line in Fig. 1 shows this assessed number in the years 1937–48 inclusive, taking 4 cases per 1,000 live births as a basis. The Registrar-General also makes a return of the annual notified deaths from pyloric stenosis for England and Wales. This appears in Fig. 1 as a continuous line.

In Fig. 2, continuous line, the annual pyloric stenosis deaths are expressed as a percentage of the assessed incidence of pyloric stenosis in England and Wales for the years 1935–48—the last year for

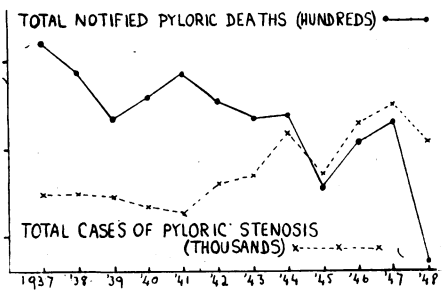

FIG. 1.

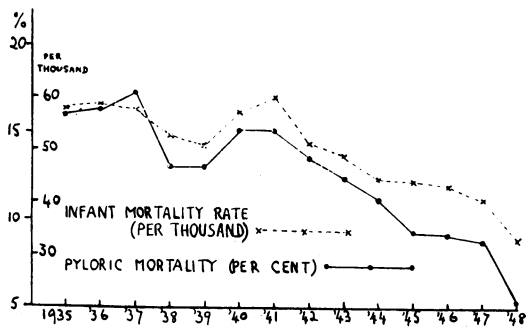

FIG. 2.

which figures are available. It will be seen that though steady improvement has been going on since 1941, until that year the death rate was well over 13%, and that not until 1948 was there any appreciable improvement on Ramstedt's figures for the five years 1929–33.

Now, there has been no great innovation in either surgical or medical treatment of the condition itself in the last twelve years, nor any great improvement in pre- or post-operative management. There has, however, been a steady decline, temporarily interrupted by the war years, in the Infant Mortality Rate. Fig. 2, dotted line, shows the curve of the infant mortality rate per 1,000 live births, set alongside that for the death-rate from pyloric stenosis. The similarity of the two curves is striking, and it is tempting to conclude that the same factors influence both mortality rates rather than that any particular details of medical or surgical treatment greatly alter the results in pyloric stenosis. This conclusion is, of course, not new. Ever since Robert Hutchison (1910) pointed out that the mortality for cases treated in hospital was many times higher than for those treated in private practice, it has been repeatedly stressed that hospital infection is the real enemy of the infant suffering from pyloric stenosis.

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Mr. R. B. Zachary: Several factors have been suggested as contributing to the success of operative treatment: persistence with breast feeding, the avoidance of parenteral fluid, the use of electrolyte therapy, and of local anæsthesia. Yet in the Children's Hospital in Boston, Mass., where the figures compare favourably with those of any other centre; these factors do not obtain.

The breast-fed baby is a rarity, general anæsthesia is the rule, and it is given by nurse anæsthetists in training. Every child has some parenteral fluid therapy, usually a combination of intravenous and subcutaneous fluids. Moreover, all the operations on hospital patients are done by the resident staff including the internes, a feature which demonstrates the safety of the operative treatment even in inexperienced hands, provided a good standard technique is used.

Since coming to Sheffield I have abandoned general anæsthesia in favour of infiltration with 0·5% local anæsthetic after sedation with 7 to 10 grains of chloral hydrate. I now feel sure that local anæsthesia is better. The ill child will stand the procedure, pre-operative therapy is needed less often, the post-operative course is smoother, and babies are on full feeds and ready for home on the third day.

Dr. J. H. Moseley: I would like to emphasize the value of rectal saline in the post-operative care of these babies. They are usually passing few stools and could be easily re-hydrated or have their chlorides restored in this way. With breast-fed babies I aim at returning them to breast feeding twenty-four hours after operation.

Dr. J. J. Kempton: There seems to be much to be said for combined responsibility in treatment. The condition is one of feeding difficulty—with a mechanical factor—and the general surgeon is unlikely to have any special interest in the problems of baby feeding. In such a system, the pædiatrician is responsible for diagnosis and pre-operative preparation. The surgeon, if he accepts the diagnosis and thinks the case suitable, operates; and the return to normal feeding in the shortest possible time is again the responsibility of the pædiatrician, every effort being made to get the baby home by the fifth post-operative day.

Dr. R. E. Bonham Carter: The pharmacology of Eumydrin is not completely understood. The pylorus is more responsive to changes of pH than to any other influence and I suggest that Eumydrin really acts indirectly by altering gastric secretion and hence the acidity of gastric juice. I wonder whether the line of treatment suggested by this idea had been explored.

Dr. D. MacCarthy: I have often observed visible peristalsis after Ramstedt's operation has been performed, which seems to indicate that the stomach is still acting in an abnormal way. I have had no experience of what happens on Eumydrin and I should like to ask the experts if visible peristalsis is abolished.

Dr. Harold Waller: I have many times observed typical gastric peristalsis and projectile vomiting in the first fortnight of life, and their disappearance under treatment with Eumydrin.

Mr. Ralph H. Gardiner (Aylesbury): I would draw attention to accidental perforation of the duodenal mucosa when the hypertrophic muscle is divided at operation. Provided that the perforation is recognized at the time and adequately dealt with, no ill-effects should result. Suture of the delicate mucous membrane is difficult, as the stitches tend to cut out, and I, personally, use a reinforcing stitch through the seromuscular coats. The stitch, although it appears to reunite the muscle, does not in my experience in any way detract from the success of the operation, and is an added safeguard. With regard to the choice of anæsthetic, I always use a local (0·5% novocain), and I think it better for the surgeon to give this himself, as, by so doing, the necessary time-interval to allow the anæsthetic to work is assured, gowns and gloves being changed before towelling up and proceeding to the actual operation.

Dr. J. H. Burkinshaw: What happens to the breast feeding when cases on medical treatment are retained in hospital for twenty days or more? The somewhat longer period of hospital stay in the Eumydrin cases seems on this account to be a point strongly against medical treatment.

Dr. Harold Waller: If the method of expressing the breast milk is correctly taught the supply can be maintained long enough in such cases.

[*May* 25, 1951]

MEETING AT THE WESTMINSTER CHILDREN'S HOSPITAL, VINCENT SQUARE, S.W.1

Laurence-Moon-Biedl Syndrome.—J. G. MILLICHAP, M.D., M.R.C.P. (for CHARLES F. HARRIS, M.D., F.R.C.P.).

S. N., aged 4 years. British.

Birth weight 7 lb. 11 oz. Obesity dating from 5 months of age. Night blindness. Spasmodic bronchitis.

Family history.—Father and paternal grandfather—diabetes mellitus. Sister died at birth with polydactyly and other abnormalities. Brother aged 11 years is normal. Cousin (child of father's twin brother) mentally defective and has double athetosis. Maternal cousins all obese. Paternal grandparents related.